Occlusion, Orthodontic Treatment, and Temporomandibular Disorders: A Review

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A review of the current literature regarding the interaction of morphologic and functional occlusal factors relative to TMD indicates that there is a relatively low association of occlusal factors in characterizing TMD. Skeletal anterior open bite, overjets greater than 6 to 7 mm, retruded cuspal position/intercuspal position slides greater than 4 mm, unilateral lingual crossbite, and five or more missing posterior teeth are the five occlusal features that have been associated with specific diagnostic groups of TMD conditions. The first three factors often are associated with TMJ arthropathies and may be the result of osseous or ligamentous changes within the temporomandibular articulation. With regard to the relationship of orthodontic treatment to TMD, the current literature indicates that orthodontic treatment performed during adolescence generally does not increase or decrease the odds of developing TMD later in life. There is no elevated risk of TMD associated with any particular type of orthodontic mechanics or with extraction protocols. Although a stable occlusion is a reasonable orthodontic treatment goal, not achieving a specific gnathologically ideal occlusion does not result in TMD signs and symptoms. Thus, according to the existing literature, the relationship of TMD to occlusion and orthodontic treatment is minor. Signs and symptoms of TMD occur in healthy individuals and increase with age, particularly during adolescence; thus, TM disorders that originate during various types of dental treatment may not be related to the treatment but may be a naturally occurring phenomenon.
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Occlusion is cited as one of the major etiologic factors within the acknowledged multifactorial origin of temporomandibular disorders (TMD).[1] This emphasis on occlusion is carried over to the most recent US Medicare guidelines, which list "malocclusion" as one of the covered temporomandibular joint (TMJ) diagnoses,[2] implying that the occurrence of occlusal variation is itself a disease. Despite much recent debate that suggests a more limited role for occlusal factors in TMJ pain and dysfunction, the question remains open for many in the field.[3]

The assumed strong association between TMD and occlusion has been a major reason that the diagnosis and treatment of these disorders has remained within the purview of dentistry. Numerous etiologic and therapeutic theories are based either partly or completely on this presumed connection and have justified many of the most common treatment approaches such as occlusal appliance therapy, anterior repositioning appliances, occlusal adjustment, restorative procedures, and orthodontic/orthognathic treatment.

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Conversely, many types of dental interventions, including routine orthodontic treatment, have been alleged to be causes of TMD.

Despite agreement among TMD experts that occlusion actually only has a relatively small role in the etiologically diverse and multifactorial origins of TMD, the influence of occlusion continues to be greatly overrated in comparison by practicing dentists and specialists outside the TMD expert circle.[4] This considerable discrepancy between the opinions of practicing dentists and TMD experts on the role of occlusion in the pathophysiology of TMD has a great impact on the contemporary quality of diagnosis and treatment for these chronic conditions. The purpose of this article is to correct occlusal misconceptions about TMD and orthodontics/orthognathic treatment maintained by popular beliefs that are not sustained in current literature, and to put occlusion into its proper perspective relative to current knowledge about its role in TMD.

Occlusal Factors and TMD

Numerous clinical studies have investigated the relationship of occlusal factors and the signs and symptoms associated with TMD in relatively large patient and nonpatient populations. Some studies reported statistically significant associations, while others did not, and few common trends were apparent. For example, Nilner[5] examined 749 juveniles and adolescents and reported that TMD signs and symptoms were associated with centric slides and balancing-side contacts. Egermark-Eriksson and colleagues,[6] after examining a random sample of 402 children, reported that occlusal supracontacts as well as many characteristics of unusual types of occlusion (ie, anterior crossbite, anterior open bite, Class II malocclusion, Class III malocclusion) were associated with signs and symptoms of TMD. Similarly, Brandt,[7] in a study of 1,342 children, noted a positive correlation of overbite, overjet, and anterior open bite with TMD.

In contrast, other investigators have reported no such associations, including DeBoever and Adriaens[8] in 135 TMD patients, Gunn and coworkers[9] in 151 migrant children, and Dworkin and colleagues[10] upon examining 592 subjects in a health maintenance organization.

Evaluation of Previous Studies

As can be seen from the above-mentioned studies, there is no universal agreement as to the relationship of occlusal factors to TMD. These differences in findings can be explained in part by problems in study design. According to Seligman,[11] some of the problems are as follows:

Symptoms Are Not Disease States. The most common type of study used in TMD research is an investigation of symptoms. This approach is problematic because isolated symptoms are not the same as disease. Any actual association of a symptom to a specific disease state may be obscured when only isolated symptoms are monitored. For example, the report of joint clicking would not differentiate disc displacement due to osteoarthrosis from simple soft tissue internal derangement. Similarly, latent muscle tenderness to palpation may reflect problems within a specific muscle group or may be an indication of global chronic fibromyalgia. If the differences among symptoms are subtle, overlapping symptoms can mask distinguishing morphologic differences by including too many different pathologic processes in the analysis.

Lack of Differential Diagnosis. Most investigations have grouped subjects into a single disease category without differentially diagnosing each patient. Thus, often it is unclear as to which disease process is being studied. Further, many patient studies are purely descriptive and do not compare patient populations with equivalent populations of healthy individuals.

Unrepresentative Samples. In some studies, the sample population does not represent the target population, particularly with regard to age and gender. For example, it is inappropriate to extrapolate to adults with osteoarthritis or fibromyalgia findings from children who rarely appear as patients with these conditions. The sample should match the target population as much as possible, especially with regard to age and sex.

Lack of Factor Definition. The definitions of the factors being studied must be made clear in operational terms, with specific criteria established for each variable. For instance, when multiple occlusal factors are grouped together into an overall variable termed "malocclusion," it is difficult to determine exactly which factors are being investigated. A factor such as posterior crossbite in one patient must be shown to have the same impact on the analysis as does a deep overbite in another patient. And if the efficacy of poorly defined occlusal treatments is examined (eg, occlusal equilibration) and the treatment is focused on the correction of a wide range of occlusal conditions rather than on the elimination of a single condition (eg, slides between centric occlusion and centric relation), the interpretation of the results of the treatment will be difficult.

Multifactorial Analysis Not Used. Combinations of factors must be studied together in a multifactorial analysis, rather than separately.[11] Isolated pairwise or sensitivity-specificity analyses attribute either major responsibility or no significant role to the occlusal factors that they examine. It is obvious that individual occlusal factors do not act in isolation from one another, and to suggest otherwise is inappropriate. With multiple factor analysis, an estimate can be made of the relative contribution of each factor in characterizing the patient.

Inappropriate Groupings of Data. Every attempt should be made to consider continuous variables over the entire range of their occurrence. Otherwise there may be an artificial or arbitrary skewing of the results. Further, the transformation of real data to unvalidated severity scales should be avoided. If a transformation is to be performed, the individual measures in the severity scale must be shown to be roughly equivalent. For example, the number of muscles tender to palpation can be quantified. To deem this information useful, it must be shown that a certain number of tender muscles is of greater concern than another number, and that there is no threshold of a minimum number of muscles before an effect is noted.

If a number of unrelated symptoms are included in a severity scale (eg, clicking, crepitus, muscle tenderness), the investigator must prove that the weighted input ascribed to each variable is valid. In addition, if one sign or symptom is emphasized in a given scoring system (eg, muscle tenderness over clicking), this preference for one type of factor also must be shown to be valid.

Conclusions. The observations of Seligman[11] illustrate the necessity of examining previous studies not necessarily on the basis of the conclusions stated by the authors, but rather by the groups studied, the criteria used, and the methods of analysis employed.

Critical Reviews of the Literature

Two of the most comprehensive reviews that have considered the relationship of occlusion to TMD have been published by Seligman and Pullinger, one considering morphologic occlusal relationships[12] and the second functional occlusal relationships.[13] These reviews were compiled in an attempt to determine consensus on the roles of various occlusal factors on the pathophysiology of TMD. These investigators considered only original research articles and emphasized those that used appropriate methodology, in particular, research that evaluated diagnostic groups or disease states rather than symptoms. The reader is referred to these articles for an in-depth literature review on each subject.

Morphologic Occlusal Relationships. Seligman and Pullinger[13] evaluated five identifiable factors related to the static occlusion.

Overbite/Open Bite. The vertical overlap of the teeth should be considered as a continuous variable. Large overbite is common in nonpatient populations, and thus this variable cannot be used to define a patient population. Studies that do not consider overbite as a continuous variable report mixed results, with a majority reporting no or very selective associations. If overbite is considered as a continuous variable, there is consensus that minimal overbite in adults is associated with osteoarthrosis. A reduced overbite may be a result of osseous changes in the joint, rather than vice versa. Skeletal anterior open bite is of particular significance. This condition is characterized as a negative vertical overlap of the anterior teeth that often is combined with occlusal contacts only in the molar region. Skeletal open bite is not common in asymptomatic nonpatients and usually is associated with disease states demonstrating intracapsular changes (eg, osteoarthrosis). Larnheim and coworkers[14] among others have noted that these occlusal changes may be a result of, rather than the cause of, these osseous changes. Skeletal anterior open bite in adults should be distinguished from anterior open bite in children, as the latter may arise from different causes (eg, thumb sucking, abnormal tongue posture).

Overjet. The horizontal overlap of the teeth does not seem to be associated with TMJ symptoms or disease. Seligman and Pullinger[13] note one exception, namely the higher prevalence of large overjet in patients with osteoarthropathies of the TMJ. Pullinger and Seligman[15] found that although larger overjets were associated with osteoarthrosis patients having a prior history of disc derangement, no such association was evident in derangement patients without osteoarthrosis. Despite the association with osteoarthrosis, large overjet is common in nonpatient populations as well, and thus this measure lacks specificity in defining patient groups.

Crossbite. Most previous studies of crossbite have considered younger patient populations.[16–18] Although asymmetric muscle activity has been reported in children with unilateral posterior crossbite,[19,20] there is little evidence that this type of morphologic relationship leads to TMJ symptomatology.[21,22] Most patient studies report no greater

prevalence of crossbite in patients as compared to studies of nonpatients.[23,24] Crossbites persisting in adults typically are skeletal in origin and do not appear to provoke TMD symptoms or disease. Thus, the correction of crossbites in adults to prevent potential TMD problems does not seem warranted.

Posterior Occlusal Support. Loss of posterior tooth support has been associated with osteoarthrosis,[25,26] but this association becomes questionable when the evaluation is controlled for age effects.[27] Research on this topic, however, is scant with regard to other patient populations. One of the few studies to consider the longitudinal relationship of the loss of posterior teeth to the health of the masticatory system has been conducted by Käyser[28] and Witter.[29] They have shown over the years that the adaptive capacity of the masticatory system is great, and that most people with loss of molar support have acceptable masticatory function and no increased amount of TMD signs and symptoms. Thus, no conclusions can be drawn regarding the benefits of prosthetically replacing missing posterior teeth as a preventative measure for TMD.

Asymmetric Contact in Retruded Cuspal Position. If imbalances of tooth contacts exist in retruded cuspal position (RCP)/centric relation, they may be most obvious in younger patient populations,[6] and as with a loss of posterior dental support, may be associated with age. No associations of this type of disorder and TMD have been reported in older populations. Prophylactic adjustment of the natural occlusion is not indicated on the basis of published studies, but the establishment of bilateral contact in RCP may be a prudent restorative goal.

Functional Occlusal Relationships. Seligman and Pullinger[12] reviewed similar published research concerning the relationship of the functional movements of the mandible to TMD.

Balancing and Working Occlusal Contacts. Most controlled surveys fail to demonstrate any association between occlusal supracontacts and TMD signs or symptoms in symptomatic nonpatients or in populations of TMD patients. Occlusal supracontacts are so common and variable[30] that they lack the sensitivity and specificity for defining a present or potential TMD population. Further, a precise and reproducible method for determining the presence of occlusal supracontacts does not exist.

Slides Between Centric Occlusion and Centric Relation. According to Seligman and Pullinger,[12] the majority of past research reports little association between the length of the slide between RCP/centric relation and intercuspal position (ICP)/centric occlusion and signs or symptoms of disorders in asymptomatic individuals. Studies of patients with radiographically determined osteoarthrosis report longer slides in arthrosis patients than in controls,[31,32] a finding that indicates that osseous remodeling or condylar lysis can be accompanied by an increased slide. In none of the studies is the amount of the slide handled as a continuous variable, thus adding bias to the interpretation of the data.

Occlusal Guidance Pattern. While there is evidence that occlusal guidance patterns can alter muscle activity levels,[33,34] there is little evidence to suggest that a given guidance pattern can provoke TMD symptomatology. Little is known concerning the role of specific guidance patterns in particular patient populations.

Parafunction. Bruxism and clenching often are cited as etiologic factors in the development of TMD, but similar to occlusal interferences, these activities (especially bruxism) seem to be endemic in the general population.[13] Furthermore, comparisons of groups identified according to self-reports of parafunctional activities are suspect because of the universality of this activity and the lack of definition as to the quantification of severity measures. Seligman and Pullinger[12] state that there is increasing evidence that parafunction is not associated with chronic occlusal factors, and thus reversible rather than nonreversible treatment should be provided in attempts to prevent or minimize possible harmful effects of this activity.[35]

Dental Attrition. There is no evidence from most nonpatient studies that dental attrition is associated with signs or symptoms of TMD. Men show greater attrition severity than women, yet they have fewer TMD symptoms. Once again, patients with osteoarthrosis have the most notable occlusal changes, often demonstrating advanced rates of attrition. These changes may be secondary to the occlusal changes resulting from the arthrosis.

Multiple Analysis of Occlusal Factors

The studies cited above considered the significance or nonsignificance of occlusal factors relative to TMD as isolated factors. Pullinger and colleagues[36] used a blinded multifactorial analysis to determine the weighted influence of each factor acting in combination with the other factors. The interaction of the following 11 occlusal factors[36] was considered in randomly collected but strictly defined diagnostic groups compared to asymptomatic controls:

1. Anterior open bite
2. Maxillary lingual posterior crossbite
3. RCP-ICP slide length
4. RCP-ICP slide asymmetry
5. Unilateral RCP contact
6. Overbite
7. Overjet
8. Dental midline discrepancy
9. Number of missing posterior teeth
10. First molar relationships (the greater of the mesiodistal maxillary discrepancies at the first molar location)
11. Right versus left first molar position asymmetry

The following are the diagnostic groups of Pullinger and coworkers[36]:

1. Disc displacement with reduction (n = 81)
2. Disc displacement without reduction (n = 48)
3. TMJ osteoarthrosis with disc displacement history (n = 75)
4. Primary osteoarthrosis (n = 85)
5. Myalgia only (n = 124)
6. Asymptomatic normals (n = 147)

The asymptomatic control subjects were considered the gold standard because they were without signs and symptoms and had no history of TMD. The samples were demographically representative, and the occlusal factors studied were collected blindly and were strictly defined. A multiple logistic regression model was used for simultaneous assessment of the relative odds of each potential occlusal factor. The outcome was always the disease classification versus the asymptomatic control subjects.

To control for age and gender, possible associations with each continuous occlusal variable were tested using the regression analysis and nominal variables by an unpaired *t* test. Of the 22 possible associations, only four were significant, and three of the four variables (overjet being the only exception) were not contributing factors in differentiating patients from controls. Thus, genders and ages were combined in this analysis.

Findings in Healthy Subjects. Wide variations in occlusal features were noted in the asymptomatic control group, including overjet from –1 to 6 mm, overbite from –2 to 10 mm, midline discrepancies to 5 mm, anteroposterior molar relationships from –6 to 6 mm, molar asymmetries from 0 to 6 mm, and RCP-ICP slides up to 2 mm in length. In addition, a wide variety of crossbites, asymmetric slides, retruded posterior contacts, and severe attrition facets were observed. Skeletal anterior open bite relationships were not observed. Thus, variations in occlusal morphology are the norm in healthy individuals, indicating the capacity of the human masticatory system to adapt to a wide variety of morphologic and functional features.

Pullinger and coworkers[36] proposed a new definition of "normal" within the context of TMD, that being those occlusal features that exist without significant elevated risk of disease. Such "normal" features include RCP-ICP slides of 2 mm or less, deep overbite, minimal overjet, midline discrepancies, all Angle classifications of occlusion, unilateral RCP contacts, and less than five missing posterior teeth. These factors alone cannot define either TMD patients or asymptomatic individuals.

Findings in Patient Populations. No single occlusal factor was able to differentiate patients from healthy subjects. There were four occlusal features, however, that occurred mainly in TMD patients and were rare in asymptomatic individuals: the presence of a skeletal anterior open bite, RCP-ICP slides of greater than 2 mm, overjets of greater than 4 mm, and five or more missing and unreplaced posterior teeth. Unfortunately, all of these signs are not only rare in healthy individuals, but also in patient populations, indicating limited diagnostic usefulness of these features.

Pullinger and coworkers[36] concluded that many occlusal parameters that traditionally were believed to be influential contribute only minor amounts to the change in risk in the multiple factor analysis used in their study. They reported that although the relative odds for disease were elevated with several occlusal variables, clear definition of disease groups was evident only in selective extreme ranges and involved only a few subjects. Thus, they concluded that occlusion cannot be considered the most important factor in the definition of TMD.

Pullinger and colleagues[36] noted, however, that the results of their study indicated that occlusal factors do contribute to TMD. Combinations of two to five of the occlusal parameters, involving eight of the 11 factors, contributed to risk for disease. These investigators stated that more commonly used statistical methods, such as robust pairwise testing, would have ignored some of these variables. The minor elevation in odds ratio revealed by the multiple factor analysis indicates that specific occlusal factors are making some biologic contribution and thus cannot be ignored. They state further that a biologic system must adapt to its various morphologic features until stability is achieved, and some occlusal features may place greater adaptive demands on the system. While most individuals compensate without problems, adaptation in others may lead to a greater risk of dysfunction.

Some occlusal differences between diagnostic groups were reported.[36] For a clinically perceptible influence to be significant, Pullinger and coworkers[36] stated that an occlusal feature would need to at least double the risk of disease (at least a 2:1 mean odds ratio). Only five occlusal conditions reached this threshold:

Anterior Open Bite. The highest odds ratio was for anterior open bite, and this occlusal manifestation was seen predominantly in both the osteoarthrosis and the myalgia-only groups, an observation noted previously by Seligman and Pullinger[13] and Stegenga.[37] For anterior open bite to be shown as an etiologic factor in the development of osteoarthritis, some evidence of this occlusal factor should exist in other diagnostic groups thought to be conditions often preceding osteoarthrosis. However, anterior open bite was not common in disc displacement disorders, with or without reduction. Further, Pullinger and coworkers[36] noted that most osteoarthrosis and myalgia patents did not present with anterior open bite.

Overjets Greater Than 6 to 7 mm. Overjets of greater than 4 mm were associated with the likelihood of osteoarthrosis, the same disease groups as the anterior open bite populations. There was no contribution to the TMJ derangement patients. Pullinger and coworkers[36] stated that some large overjets in adults can be secondary to the condylar repositioning seen with advanced osteoarthrosis. An overjet of 6 mm or larger was needed for a subject to be assigned to one of these disease classifications with an odds ratio of at least 2:1. The occurrence of a progressively increasing overjet in adults should alert the clinician to evaluate a patient for other signs of TMD disease.

RCP-ICP Occlusal Slides. Small occlusal slides, mostly under 1 mm, were common in all patient groups and normals, but sagittal slides longer than 2 mm were found in the disease groups only. None of the asymptomatic subjects had occlusal slides greater than 2 mm, and only 6% had slides longer than 1 mm. Pullinger and coworkers[36] found that larger slides occasionally were associated with degenerative changes within the TMJ. A slide of 5 mm or greater would be necessary to reach a 2:1 odds ratio threshold for notable risk, and this ratio never was observed in the patients. Thus, the effective clinical contribution of this factor was determined to be minimal.

Because an occlusal slide has not been shown to be a contributor to the TMD equation, the prophylactic elimination of most slides through clinically relevant occlusal equilibration procedures is not indicated. Even in the presence of what may appear to be symptoms associated with an occlusal slide, the removal of a large discrepancy between centric occlusion and centric relation may not be advisable because the slide may be a consequence of an articular disorder (eg, primary arthrosis) rather than as a result of occlusal factors. It should be noted that the above three factors that have emerged from the multiple factor analysis have a primary association with osseous and ligamentous changes within the articular compartments of the temporomandibular joints. These occlusal factors may in fact be a result of, rather than a cause of, these joint changes.

Unilateral Maxillary Lingual Crossbite. This occlusal feature, occurring in about 10% of the adult population, has a greater risk for assignment to the TMJ derangement groups. Nearly one fourth of the nonreducing disc displacement patients included this feature, and the odds ratio that an individual with this type of crossbite also would have TMJ disc displacement with reduction was over 3:1.[36] Similar odds ratios were seen for the disc displacement group without reduction (2.6:1) and also in the osteoarthrosis with disc displacement history group (1.96:1). Pullinger and coworkers[36] note that the persistence of an odds ratio for disease association into adulthood indicates that the adaptive response in a small percentage of subjects may be less than optimal and leads to the suggestion that functional adaptation to a unilateral posterior crossbite in childhood may be made at the expense of the articular disc through the development of internal derangement, including a few that eventually progress to arthrosis. These investigators believe that a case can be made for the treatment of children with unilateral crossbites to reduce the adaptive demands on the masticatory system. Conversely, the orthodontic correction of unilateral crossbite in adults to prevent TMJ derangement development probably is not warranted, because skeletal adaptation already has occurred.

Missing Posterior Teeth. In the samples studied by Pullinger and coworkers,[36] extensive posterior tooth loss was not common. Five or more posterior teeth needed to be missing before the odds ratio of assignment to disease groups assumed a minimal critical ratio of 2:1 for osteoarthrosis with disc displacement history and primary osteoarthrosis and also for disc displacement with reduction. Age is associated with both osteoarthrosis[38] and tooth loss,[39] indicating that the increase in odds ratio in patients with osteoarthrosis and more than four missing teeth also may be a reflection of age. Much of the increase in tooth loss in the patients characterized by disc displacement with reduction,

a group of patients that generally was younger than the osteoarthrosis groups, was premolar extraction as part of an orthodontic treatment. Pullinger and coworkers[36] noted that the contribution of the extraction of two to four teeth per se, for example, as part of an orthodontic treatment protocol, was negligible in most cases when other variables were controlled. As mentioned earlier, longitudinal studies of patients with multiple missing posterior teeth have shown acceptable masticatory function without increased signs and symptoms of TMD.[28,29]

Conclusions. The multifactorial analysis of Pullinger and coworkers[36] has shown that, except for a few defined occlusal conditions, there is a relatively low risk of occlusal factors associated with TMD. In a subsequent reanalysis of these data, Seligman[11] has estimated that overall contribution of occlusal factors in defining TMD patients probably is from 10% to 20%, which leaves 80% to 90% of the TMD patient characteristics unexplained by their occlusion. None of these studies can identify a cause and effect relationship of occlusal factors to TMD. However, the fact that the correlation coefficients usually are in the .3 range explains less than 10% of the variation. In a specific disease state, the causative agent usually explains 80% to 90% of the variation.

Orthodontic Treatment and TMD

Although long recognized by orthodontists as a clinical problem, little emphasis was placed on the diagnosis and treatment of TMD within the specialty until about the mid-1980s. Traditionally, scant mention was made of TMD treatment in the curricula of graduate programs in orthodontics, and only cursory examinations of the TMJ region were conducted in routine orthodontic clinical examinations.

However, the interest of the orthodontic community was awakened abruptly in the late 1980s following litigation that alleged that orthodontic treatment was the proximal cause of TMD in orthodontic patients, with substantial monetary judgments being awarded to several plaintiffs.[40] This litigious climate stimulated the American Association of Orthodontists not only to sponsor a series of risk management teleconferences and newsletters, but also to underwrite research concerning the relationship of orthodontic treatment to TMD. This series of clinical studies, the results of which were published in the January 1992 issue of the *American Journal of Orthodontics and Dentofacial Orthopedics*, reported that orthodontic treatment generally is not a primary factor in TMD. Yet, this controversy is not settled, as is indicated by the recent viewpoint article of Thompson[41] that once again cites faulty intercuspation of the teeth and dental intrusions into the freeway space as two of the many etiologic factors that may lead to TMJ dysfunction and its sequelae.

Review of the Literature

Prior to 10 years ago, surprisingly few methodologically sound clinical studies regarding the relationship between orthodontic treatment and TMD had been published. In a comprehensive review of the literature on this subject that was published between 1966 and 1988, Reynders[42] divided 91 publications into three categories: viewpoint articles, case reports, and sample studies. The most numerous were viewpoint articles (n = 55), publications that usually were anecdotal in nature, stating the opinion of the author regarding the orthodontic-TMD relationship. Little (or more commonly no) data were presented to support the opinion. Further, Reynders[42] notes that 23 of the 55 viewpoint articles were published in *The Functional Orthodontist*, with articles advancing the concepts that orthodontic treatment can either cause or cure TMD. The second most frequent type of article (n = 30) was the case report, a category of publication that described the influence of certain orthodontic treatment modalities used in one or more patients on the signs and symptoms of TMD. The least numerous (n = 6) were in the third category of sample studies, investigations that reported data from large sample groups. These studies were of variable quality, often having the same methodologic problems and limitations as discussed previously for studies of occlusal factors. Since 1988, a substantial number of clinical investigations have considered the association of orthodontics and TMD (Table 1).

Viewpoint articles, of course, are not suitable for critical evaluation of associations between two entities such as orthodontic treatment and TMD; they are, however, useful in identifying questions that may be worthy of scientific investigation. Some of these questions are as follows:

1. What is the prevalence of signs and symptoms of TMD in orthodontically untreated populations?
2. Does orthodontic treatment lead to a greater incidence of signs and symptoms of TMD?

Table 1 Major Studies of the Relationship Between Orthodontic Treatment and Signs and Symptoms of TMD

Author	Sample	Appliance	Tooth extraction	Relationship
Sadowsky and BeGole, 1980[50]	75 treated 75 untreated	Fixed	No	No
Larsson and Rönnerman, 1981[53]	23 treated	Fixed	No	Improvement
Jandon anf Hasund, 1981[67]	60 treated 30 untreated	Fixed Functional	Yes	Improvement
Sadowsky and Polson, 1984[51]	207 treated 214 untreated	Fixed	No	No
Pancherz, 1985[68]	22 treated	Functional	No	No
Dibbets and van der Weele,1987[64]	135 treated	Fixed (72) Functional (63)	Yes	No
Dahl et al, 1988[55]	51 treated 47 untreated	Fixed Functional	No	No
Smith and Freer, 1989[57]	87 treated 28 untreated	Fixed	No	No
Sadowsky et al, 1991[76]	160 treated	Fixed	Yes	No
Dibbets and van der Weele, 1992[66]	92 treated	Fixed Functional	Yes	No
Luecke and Johnston, 1992[83]	42 patients	Fixed	Yes	No
Artun et al, 1992[85]	63 treated	Fixed	Yes	No
Kremenak et al, 1992a[58]	65 treated	Fixed	Yes	No
Kremenak et al, 1992b[59]	109 treated	Fixed	No	No
Egermark and Thilander, 1992[100]	402 mixed	Fixed Functional	No	Improvement
Paquette et al, 1992[77]	63 orthodontic patients	Fixed	Yes	No
Luppanapornlarp and Johnston, 1993[79]	62 orthodontic patients	Fixed	Yes	No
Beattie et al, 1994[78]	63 orthodontic patients	Fixed	Yes	No

3. Does the type of appliance (eg, fixed versus functional; orthodontic versus orthopedic) make a difference?
4. Does the removal of teeth as part of an orthodontic protocol lead to a greater incidence of TMD?
5. Can orthodontic treatment lead to a posterior displacement of the mandibular condyle?
6. Should the occlusions of orthodontic patients be treated to specific gnathologic standards?
7. Does orthodontic treatment prevent TMD?

Although the literature is not as extensive on the relationship of orthodontics to TMD as it is to the occlusal/TMD relationship, the questions outlined above have been addressed in a substantial number of recent studies. These reports are discussed in detail below, with many of the investigations considering more than one question.

Occurrence of Signs and Symptoms of TMD in Healthy Individuals. We previously have seen the importance of studying healthy asymptomatic populations in assessing the relationship of occlusal factors to TMD. Such is the case when orthodontic populations are considered.

Numerous epidemiologic studies have examined the prevalence of signs and symptoms associated with TMD in a wide variety of subject populations (Table 2). In general, the prevalence has been shown to be statistically significant, with an average of 32% reporting at least one symptom of TMD, and an average of 55% demonstrating at least one clinical sign.

Cross-sectional epidemiologic studies of specific adult nonpatient populations indicate that at any given time, between 40% and 75% have at least one sign, and about one third report at least one symptom of TMD.[43–45] According to Montegi and coworkers,[46] the point prevalence of symptoms in children and teenagers is lower, about 12% to 20%.

Because of the longitudinal nature of orthodontic treatment (eg, 2 to 3 years for adolescents; 5 to 7 years for patients starting a two-phase treatment protocol in the early mixed dentition), an understanding of the changes in the signs and symptoms of TMD in a healthy population is essential. Several investigators have noted that signs and symptoms of TMD generally increase in frequency and severity, beginning in the second decade of life.[39,47,48] Wänman and Agerberg[49] have noted that

Table 2 Epidemiologic Studies of the Signs and Symptoms of TMD in Untreated Populations

Author	No. of individuals	No. of females/males	Age (years)	Population	Prevalence: At least one symptom	Prevalence: At least one clinical sign
Nilner and Lassing, 1981[101]	440	218/222	7–14	Swedish children	36	72
Egermark-Eriksson et al, 1981[100]	136	74/62	7	Swedish children	39	33
	131	61/70	11	Swedish children	67	46
	135	59/76	15	Swedish children	74	61
Gazit et al, 1984[102]	369	181/188	10–18	Israeli children	56	44
Nilner, 1986[5]	309	162/147	15–18	Swedish children	41	77
Swanljung and Rantanen, 1979[103]	583	341/256	18–64	Finnish workers	58	86
Solberg et al, 1979[104]	739	370/369	19–25	American university students	26	76
Pullinger et al, 1988[21]	222	102/120	19–40	Dental hygiene and dental students	39	48
Rieder et al, 1983[105]	1,040	653/387	13–86	American private practice	33	50
Ingervall et al, 1980[106]	389	0/389	21–54	Swedish reservists	15	60
Osterberg and Carlsson, 1979[107]	384	198/186	70	Swedish retirees	59	37
Agerberg and Inkapööl, 1990[108]	637	323/314	18–64	Swedish adults	14	88
De Kanter et al, 1993[45]	3,468	1815/1653	15–74	Dutch nationals	22	45
Magnussen et al, 1993[99]	293	164/129	17–25	Swedish young adults	43 (only)	
Glass et al, 1993[109]	534	317/217	18–65	Kansas City adults	46 (only)	
Tanne et al, 1993[110]	323	146/86	3–29	Prospective orthodontic patients	16	15
Totals*:	n = 10,032 (symptoms) n = 9,205 (signs)				n = 3,254 = 32%	n = 5,023 = 55%

Adapted and expanded from Okeson.[35]
*The numbers of subjects with symptoms and clinical signs were determined for each study by multiplying the total number of subjects by the percentages of subjects exhibiting at least one symptom and at least one clinical sign. For the total number of subjects considered in the table, 32% had at least one symptom and 55% had at least one clinical sign.

the incidence of joint sounds in young adults in their late teens can be as high as 17.5% over a 2-year period. Thus, the occurrence of joint sounds during orthodontic treatment must be considered within the context of longitudinal changes in a comparable untreated population studied during the same time interval.

Orthodontic Treatment Versus No Treatment. Two of the first investigations sponsored by the National Institutes of Health to consider the relationship between orthodontics and TMD were initiated about 15 years ago (Table 1). These research efforts considered the prevalence of TMD and the status of the "functional occlusion" (to be discussed later) in large groups of subjects who had undergone orthodontic treatment at least 10 years previously.

Sadowsky and BeGole[50] reported on the findings from a University of Illinois study of 75 adult subjects who, at least 10 years previously, had been treated with full orthodontic appliances as adolescents. The treated group was compared to a group of 75 adults with untreated malocclusions. In a subsequent article by Sadowsky and Polson,[51] the sample from the Illinois study (increased to 96 treated and 103 controls) was compared to a treatment group of 111 subjects who had been treated at least 10 years previously at the Eastman Dental Center and a control group of 111 individuals with untreated malocclusions. In the two studies, 15% to 21% of the subjects presented with one or more sign of TMD and 29% to 42% had at least one or more symptom of TMD, usually joint sounds. There were no statistically significant differences between the treated and untreated groups.[52] The results of these two studies provide evidence in support of the concept that orthodontic treatment performed during adolescence generally did not increase or decrease the risk of developing TMD later in life.

Another study of the long-term effects of orthodontic treatment was conducted by Larsson and Rönnerman.[53] They evaluated 23 adolescent patients who had been treated orthodontically at

least 10 years earlier. Eighteen of the patients were treated with fixed appliances, while five patients received activator treatment. Using the Helkimo index[54] as an evaluative tool, mild dysfunction was recorded in eight patients, while one patient had severe dysfunction. Comparing their results to published epidemiologic studies, Larsson and Rönnerman[53] stated that comprehensive orthodontic treatment can be undertaken without fear of creating TMD problems.

Dahl and coworkers[55] examined 51 subjects 5 years after the completion of orthodontic treatment. Signs and symptoms of TMD were noted and compared to the findings from a similar group of 47 untreated individuals. According to the authors, "nobody really had craniomandibular disorders" in either group. Severe symptoms (eg, difficulties in wide opening, locking, pain on mandibular movement) typically were not observed; however, mild symptoms (eg, joint sounds, muscle fatigue, stiffness of the lower jaw) were observed more frequently in the untreated group than in the treated group, a difference that was statistically significant. Dahl and coworkers[55] noted that the number of subjects in both groups who had at least one mild symptom was relatively high (70% in the treated group, 90% in the untreated group), especially in comparison to the previously mentioned investigation of Larsson and Rönnerman,[53] which reported a 27% occurrence of mild dysfunction in their treated patients. They reported that differences between samples may be due as much to measuring differences (eg, lack of factor definition, differences in the interpretation of the criteria of the Helkimo index) as to a true reflection of differences between groups.

Rendell and colleagues[56] examined 462 patients receiving treatment in an orthodontic graduate clinic (90% adolescents, 10% adults), using a modification of the Helkimo[54] index. Eleven of the patients presented with TMD signs/symptoms prior to treatment. During the 18-month study period, none of the patients who had been free from signs/symptoms at the beginning of treatment developed signs or symptoms of TMD. No clear or consistent changes in the levels of pain and dysfunction occurred during the treatment period in those patients with preexisting signs or symptoms. Rendell and coworkers[56] concluded that a relationship could not be established in their patient population between orthodontic treatment and either the onset or the change in severity of TMD signs and symptoms.

One of the few clinical studies to report positive findings is the investigation of Smith and Freer,[57] which examined 87 patients treated with full orthodontic appliances in adolescence. About two thirds of the sample had permanent teeth removed as part of the treatment protocol. The treated group was compared to an untreated control group of 28 individuals. Four years following the end of retention, symptoms were found in 21% of the treated group and 14% of the control subjects, a difference that was not significant statistically. However, the investigators noted a single sign that was statistically significant, the exception being the association between what they termed "soft clicks" and previous treatment. Soft clicks were found in 64% of the treatment group and 36% of the untreated group. They, however, did not find any difference in joint sounds (ie, crepitus as determined by stethoscopic examination) between the two groups. Interestingly, the authors concluded the article by stating: "The null hypothesis that there is a significant association between orthodontic treatment and occlusal or joint dysfunction has been rejected by nearly all previously reported studies and continues to be rejected by the present study."

There have been relatively few prospective studies that have examined the relationship of orthodontics to TMD. The two major investigations have been conducted at the University of Groningen in the Netherlands (to be discussed later) and at the University of Iowa.[58–60] In the latter ongoing study, 30 new orthodontic patients have been enrolled annually since 1983. The method of Helkimo[54] has been used to collect TMD data prior to orthodontic treatment and at yearly intervals following the completion of treatment. Patients were treated using comprehensive edgewise appliances with and without extractions. No longitudinal data on a comparable untreated population were obtained.

Kremenak and coworkers[59] have reported data from pretreatment and posttreatment examinations from 109 patients. Data on follow-up examinations from 1 to 6 years posttreatment were available on declining samples sizes of 92, 56, 33, 19, 11, and 7 individuals. No statistically significant differences were noted between mean pretreatment and posttreatment Helkimo scores for any of the various groupings. Ninety percent of the patients had Helkimo scores that remained the same or improved, and 10% had scores that worsened (an increase from 2 to 5 Helkimo points). Kremenak and colleagues[58,59] concluded that the orthodontic treatment experienced by their sample was not an important etiologic factor for TMD.

Hirata and coworkers[61] examined 102 patients before and after orthodontic treatment for signs of

TMD. Findings from this group were compared to findings from 41 untreated subjects matched for age. The incidence of TMD signs for the treatment and control groups was not a statistically significant difference.

Type of Orthodontic Mechanics Used. In the other major longitudinal study of this subject, Dibbets and colleagues[62-66] followed 171 patients, 75 of whom were treated using the Begg technique (most patients had extractions as part of their treatment protocol), 66 patients treated using activator therapy, and 30 patients treated with chin cups. The pretreatment documentation revealed a strong dependence of the prevalence of signs and symptoms on age: from 10% at age 10 years, signs increased to 30% at 15 years, while symptoms increased to over 40%. They also noted that at the end of treatment, the fixed appliance group had a higher percentage of objective symptoms than did the functional group, but no differences existed at the 20-year follow-up.[66]

Janson and Hasund[67] conducted a similar study of adolescent patients with Class II division 1 malocclusion examined 5 years out of retention. Thirty patients underwent a two-phase treatment regimen (headgear/activator therapy followed by fixed appliances) without the removal of teeth, and 30 patients were treated using fixed appliances following the removal of four premolars. An additional 30 untreated subjects were used as controls. One or more symptoms were reported in about 42% of the subjects overall (treated and untreated), with similar findings for the clinical dysfunction index.[54]

One prospective study examined the effect of functional mandibular advancement in patients with Class II division 1 malocclusion. Pancherz[68] used the banded Herbst appliance only in 22 adolescent patients with Class II division 1 malocclusion during a treatment period of 6 months. Following an initial incisal edge-to-edge bite registration, Pancherz reported that a number of patients complained of muscle tenderness during the first 3 months of treatment. However, 12 months following treatment, the number of subjects with symptoms was the same as before treatment.

Extraction and TMD. Viewpoint articles and texts have strongly associated the extraction of premolars with the occurrence of TMD in orthodontic patients.[69-75] These articles are long on opinion and short on data.

The clinical studies that have dealt with this issue have not shown a relationship between premolar extraction and TMD. For example, Sadowsky and coworkers[76] reported findings on 160 patients, 54% of whom were treated using extraction treatment strategies. Joint sounds were monitored before and after treatment in 87 extraction patients and 68 nonextraction orthodontic patients. Before treatment, 25% of patients had joint sounds, whereas 16.5% had sounds after treatment. Similarly, 14% of patients had reciprocal clicking; only 8% had clicking after treatment. The investigators concluded that their findings did not indicate a progression of signs and symptoms to more serious problems during treatment. They also reported no increase in the risk of developing joint sounds regardless of whether teeth were removed.

The long-term effects of extraction and nonextraction edgewise treatments were compared in 63 patients with Class II division 1 malocclusions who were identified by discriminant analysis as being equally susceptible to the two treatment strategies.[77,78] In terms of a menu of 62 signs and symptoms (eg, muscle palpation, joint function) that commonly are thought to be characteristic of TMD, there were no differences between extraction and non-extraction samples. A follow-up study by Luppanapornlarp and Johnston[79] that examined an additional 62 "clear-cut" patients (those in the tail of the distribution) also noted that both extraction and nonextraction samples demonstrated similar findings.

The longitudinal studies at the University of Iowa also have addressed this question. Kremenak and colleagues[58] followed three groups of patients: 26 patients treated without extraction, 25 patients with four premolars extracted, and 14 patients with two maxillary premolars extracted. No significant intergroup differences between mean pretreatment or posttreatment Helkimo scores were noted. A small but statistically significant improvement in Helkimo scores was observed posttreatment in both the nonextraction group and the group with four extracted premolars.

Dibbets and van der Weele[65] followed 111 of the original 172 orthodontic patients in the Groningen study over a 15-year period. In this group, a nonextraction approach was used in 34% of the patients, four premolars were extracted in 29%, and other extraction patterns were used in the remaining 37%. Functional appliances were used in 39%, fixed appliances (Begg) were used in 44%, and chin cups in 17% of the patients. Symptoms increased from 20% to 62%; signs of clicking and crepitus increased from 23% to 36% after 4 years and then stabilized. In contrast to the finding from the first 10 years during which there was no difference between the three treatment groups with regard to clicking, after 15 years this symptom was

seen more often in the premolar extraction group. The authors noted, however, that clicking was higher in the premolar extraction group before treatment was started and concluded that the original growth pattern, rather than the extraction protocol, was the most likely factor responsible for the TMD complaints seen many years posttreatment. These investigators also noted that for a substantial number of patients, symptoms of TMD appeared and disappeared during the course of study. In the 20-year follow-up, the difference had disappeared completely.[66] They also noted that even though the overall incidence of symptoms increased with time, many previously symptomatic children became asymptomatic at the time of subsequent evaluations.

Finally, in the multiple factor analysis of occlusal factors described previously, Pullinger et al[36] noted that the contribution of the extraction of two to four teeth per se, for example, as part of an orthodontic treatment protocol, was negligible in most cases when other variables were controlled.

Orthodontic Treatment and Posterior Condylar Displacement. A number of viewpoint articles have asserted that a wide variety of traditional orthodontic procedures (eg, premolar extraction, extraoral traction, retraction of maxillary anterior teeth) cause TMD signs and symptoms by producing a distal displacement of the condyle.[73,80,81] This allegation is opposite to that of the gnathologist's approach to condylar position, a topic that will be considered in the next section.

Gianelly et al[82] used corrected tomograms to evaluate condylar position before orthodontic treatment in 37 consecutive patients aged 10 to 18 years and compared them with tomograms from 30 consecutively treated patients treated with fixed appliances (edgewise or Begg) and the removal of four premolars. No differences in condylar position were noted between groups. The position of the condyle tended to be centered within the glenoid fossa, but wide variation in condylar position was noted in both groups.

Luecke and Johnston[83] evaluated the pretreatment and posttreatment records of 42 patients treated with fixed appliances in conjunction with the removal of two maxillary premolars. The results of this study indicated that the majority of patients (about 70%) undergo a forward mandibular displacement and a slight opening rotation of the mandible. The remainder of the sample had distal movement of the condyle. Incisor changes were essentially unrelated to condylar displacement during treatment. Luecke and Johnston[83] stated that a change in the spatial position of the mandible is a function of changes in the anteroposterior position of the occluding buccal segments, rather than the relatively nonoccluding incisors. These observations also are supported by the findings of Tallents and coworkers.[84]

The recall studies of Paquette and coworkers[77] and Luppanapornlarp and Johnston[79] have reported no differences between groups with regard to TMD signs and symptoms. They also noted that both extraction and nonextraction treatments produced a mean mesial displacement of the mandible.

Artun and colleagues[85] also investigated the relationship of orthodontic treatment to posterior condylar displacement. Sixty-three female patients were evaluated after routine fixed appliance treatment (29 with extraction and 34 without extraction). Condylar position was measured in percent anterior and posterior displacement from absolute concentricity on the basis of sagittally corrected tomograms. The investigators did note a mean difference in condylar position between the two treatment groups, but the difference was due mainly to the occurrence of presumed anteriorly displaced condyles in the nonextraction group (data on the pretreatment position of the condyle were not obtained). They did note that the condyles in patients with clicking were in a more mean posterior position, but there was a wide variation of condylar position in all samples, and this variation also extended to different tomographic sections within the same condyle. They concluded that any posterior condylar position was not due to orthodontic treatment.

Gnathologic Principles and Orthodontic Treatment. Several viewpoint articles[86–91] have maintained that TMD may result from a failure to treat orthodontic patients to gnathologic standards that include the establishment of a "mutually protected occlusion" and proper seating of the mandibular condyle within the glenoid fossa (in contrast to the more anterior position of the condyle advocated by the so-called "functional orthodontists"). The gnathologists claim that nonfunctional occlusal contacts, when introduced through orthodontic treatment, can lead to signs and symptoms of TMD.

The discussion of the relationship of occlusion and malocclusion to TMD presented earlier in this paper illustrates the lack of association between most occlusal factors and TMD. Pullinger and coworkers[36] reported that small occlusal slides, mostly under 1 mm, are common in asymptomatic subjects as well as TMD patients. Only when a slide between RCP and ICP becomes extreme (5 mm or greater) does the odds ratio for disease

become elevated. Thus, finishing orthodontic treatment with a modest slide typically is within the adaptive capabilities of most patients.

Sadowsky and BeGole[50] and Sadowsky and Polson[51] evaluated the prevalence of nonfunctional occlusal contacts in patients at least 10 years after orthodontic treatment. They noted a high incidence of such occlusal contacts in both orthodontic and control groups. Similar findings have been reported by Cohen[92] and Rinchuse and Sassouni,[93] among others.

Although it probably is prudent to establish morphologic treatment goals that mimic what is observed in untreated occlusions that have been judged normal or ideal, such as the "six keys of ideal occlusion" advocated by Andrews,[94,95] and to treat a patient orthodontically so that there is a minimal (< 2 mm) slide between RCP and ICP, the establishment of an occlusion that meets gnathologic ideals probably is unnecessary, particularly in adolescent patients, and sometimes the attainment of a gnathologic ideal may be impossible in certain adult patients.

Orthodontic Treatment to Prevent TMD. This last topic probably is the most difficult to investigate, given the prevalence of signs and symptoms of TMD in healthy individuals and the many types of orthodontic treatment philosophies, goals, and techniques in existence today. The question of whether orthodontic treatment can prevent TMD is complicated further by many of the unsubstantiated viewpoint articles that claim preventive capabilities of nonextraction treatment, functional appliances, and some of the more nontraditional orthodontic treatment protocols (eg, second molar extraction and third molar replacement) that have been advocated vigorously.[72–74,96–98]

As discussed above, most studies that have compared treated and untreated populations have found no differences between groups in the occurrence of TMD signs and symptoms. One of the few investigations that found improved TMD health in a treated group was the sample studied by Magnusson and coworkers[99] and Egermark and Thilander.[100] These investigators reevaluated at 5 and 10 years respectively a group of 402 children and adolescents who originally had been evaluated cross sectionally by Egermark-Eriksson[16] and Egermark and Thilander.[100] The sample originally was divided into three groups according to age (7, 11, and 15 years). About one third of the sample had received orthodontic treatment at the end of the final examination period. Bruxism awareness and subjective symptoms of TMD increased in all age groups, with symptoms slightly more pronounced in untreated individuals. The investigators also noted that clicking recorded at the first examination may disappear at subsequent examinations and that clicking may appear at subsequent intervals regardless of whether the subject underwent orthodontic treatment. As in many previous studies, the Helkimo[54] index was used to measure clinical signs of TMD in the oldest age group (25 years). The clinical dysfunction index outcome was lower in those experiencing orthodontic treatment than those who had not.

As mentioned earlier, a trend toward decreased prevalence of TMD signs and symptoms in treated patients also was noted by Sadowsky and Polson[51] and Dahl and coworkers.[55] The signs and symptoms of TMD in the previously treated orthodontic patients were seldom severe enough to say that these patients suffered from TMD (even if they had signs and symptoms).

Summary

In this paper, we have attempted to review the current literature regarding the interaction of morphologic and functional occlusal factors relative to TMD. We have cited the articles of Seligman and Pullinger[12,13] as comprehensive reviews of the literature on this subject. Of particular importance is the methodologic weakness of previously published studies, particularly with regard to the sample groups studied, the criteria used for evaluation, and the method of analysis employed.

The multiple factor analysis of Pullinger and colleagues[36] has indicated that there is a relatively low association of occlusal factors in characterizing TMD. This association, however, is not zero, and several occlusal features characterized the diagnostic groups:

1. Skeletal anterior open bite
2. Overjets greater than 6 to 7 mm
3. RCP/ICP slides greater than 4 mm
4. Unilateral lingual crossbite
5. Five or more missing posterior teeth

The first three factors often are associated with TMJ arthropathies and may be the result of an osseous or ligamentous change within the temporomandibular articulation. Overall, Seligman[11] estimates that the total contribution of occlusal factors to the multifactorial characterization of TMD patients is about 10% to 20%, with other factors, both pronounced and subtle, interacting

and providing the remaining 80% to 90% of the differences between patients and healthy subjects.

The second part of this paper reviewed the current literature regarding the relationship of orthodontic treatment to TMD. Although this subject became a focus of conversation within the dental and legal communities in the late 1980s, little substantive research on this topic was available until recently.

The findings of current research on this subject can be summarized as follows:

1. Signs and symptoms of TMD occur in healthy individuals.
2. Signs and symptoms of TMD increase with age, particularly during adolescence. Thus, TMD that originates during treatment may not be related to the treatment.
3. Orthodontic treatment performed during adolescence generally does not increase or decrease the odds of developing TMD later in life.
4. The extraction of teeth as part of an orthodontic treatment plan does not increase the risk of TMD.
5. There is no elevated risk for TMD associated with any particular type of orthodontic mechanics.
6. Although a stable occlusion is a reasonable orthodontic treatment goal, not achieving a specific gnathologic ideal occlusion does not result in TMD signs and symptoms.
7. No method of TMJ disorder prevention has been demonstrated.
8. When more severe TMD signs and symptoms are present, simple treatments can alleviate them in most patients.

Thus, according to the existing literature, the relationship of TMD to occlusion and orthodontic treatment is minor. The important question that still remains in dentistry is how this minor contribution can be identified within the population of TMD patients. Future research should be directed toward developing a more complete understanding of these occlusal factors so that reliable criteria can be developed to assist the dental practitioner in deciding when dental therapy plays a role in the management of TM disorders. Reliable criteria likely would spare many TMD patients significant dental therapies and related health costs. Until such criteria are developed, the dental profession should be encouraged to manage TMD symptoms with reversible therapies, only considering permanent alterations of the occlusion in patients with very unique circumstances.

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Resumen

Revisión literaria sobre oclusión, tratamiento de ortodoncia y los desórdenes temporomandibulares

Al realizar una revisión de la literatura con respecto a la interacción de los factores oclusales funcionales y morfológicos relativos a los desórdenes temporomandibulares (DTM), se ha encontrado que hay una asociación relativamente baja en cuanto a los factores oclusales al caracterizar el desorden temporomandibular. La mordida abierta anterior esquelética, las sobremordidas horizontales mayores de 6–7 mm, la posición cuspidea retruida/los deslizamientos de la posición intercuspídea mayores de 4 mm, la mordida cruzada lingual unilateral,y 5 ó mas dientes posteriores ausentes son las cinco características oclusales que han sido asociadas con grupos de diagnóstico específicos de DTM. Los primeros tres factores a menudo están asociados con artropatías de la articulación temporomandibular (ATM) y pueden ser el resultado de camblos en los huesos o los ligamentos dentro de la ATM. En cuanto a la relación del tratamiento de ortodoncia con los DTM, la literatura actual indica que la ortodoncia efectuada durante la adolescencia generalmente no aumenta o disminuye las posibilidades de desarrollar DTM mas tarde. No existe un riesgo elevado de DTM asociados con ningún tipo particular de técnica ortodóntica o con protocolos de extracción. Aunque la oclusión estable es un objetivo razonable del tratamiento de ortodoncia, el hecho de no alcanzar una oclusión específica, gnatológicamente ideal, no quiere decir que se van a presentar signos y síntomas temporomandibulares. Por lo tanto, de acuerdo a la literatura actual, la relación de los DTM con la oclusión y el tratamiento ortodóntico, es mínima. Los signos y síntomas de los DTM ocurren en individuos sanos y aumentan con la edad, particularmente durante la adolescencia; por lo tanto, los DTM que se originan durante varios tipos de tratamientos dentales quizás no estén relacionados al tratamiento, pero quizás pueden ser un fenómeno que ocurre naturalmente.

Zusammenfassung

Oklusion, kieferorthopädische Behandlung und Myoarthropathien: Eine Literaturübersicht

Eine Übersicht der aktuellen Literatur unter Berücksichtigung der Wechselwirkung von morphologischen und funktionellen okklusalen Faktoren bezüglich der Myoarthropathien zeigt eine relativ kleine Assoziation zwischen okklusalen Faktoren und Myoarthropathien. Skelettal anterior offener Biss, Overjets grösser als 6–7 mm, eine Ableitbewegung von RK zu IK von mehr als 4 mm, unilateraler Kreuzbiss und fünf oder mehr fehlende Seitenzähne stellen die fünf okklusalen Merkmale dar, welche mit spezifischen diagnostischen Gruppen bei Myoarthropathien in Zusammenhang gebracht wurden. Die ersten drei Faktoren sind oft mit Kiefergelenksarthropathien assoziiert und könnten das Resultat einer ossären oder ligamentären Veränderung innerhalb des Gelenks darstellen. Was eine Wechselwirkung von kieferorthopädischen Behandlungen und Myoarthropathien anbelangt, zeigt die aktuelle Literatur kein erhöhtes oder erniedrigtes Risiko des Auftretens einer Myoarthropathie im Erwachsenenalter, wenn eine kieferorthopädische Behandlung in jugendlichem Alter durchgeführt wird. Es existiert kein erhöhtes Risiko für das Auftreten einer Myoarthropathie durch irgendeine kieferorthopädische Technik oder durch Extraktionen. Obwohl eine stabile Okklusion ein vernünftiges kieferorthropädisches Behandlungsziel darstelt, hat das Verfehlen einer gnathologisch idealen Okklusion keine Myoarthropathie zur Folge. Daher ist in Übereinstimmung mit der existierenden Literatur die Beziehung zwischen Myoarthropathien und Okklusion beziehungsweise kieferorthopädischer Behandlung unbedeutend. Zeichen und Symptome einer Myoarthropathie treten bei gesunden Individuen auf und nehmen mit dem Alter zu, vor allem während der Adoleszenz. Daher sind Myoarthropathien, welche während verschiedener Arten von zahnärztlicher Behandlung auftreten, nicht gezwungenermassen mit der Behandlung verbunden, sondern können ein natürlich auftretendes Phänomen darstellen.